Post-traumatic Myofascial Pain
of the Head and Neck

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Post-traumatic myofascial pain describes the majority of chronic head and neck pain seen in clinical practice. If conditions such as vascular headaches, neuropathic pain, degenerative cervical joint disease, and dental pain are excluded, myofascial tissues are directly or indirectly involved in all other forms of head and neck pain. The most common of these include temporomandibular disorders, neck pain such as whiplash-associated disorder, cervicogenic headaches, and tension-type headaches. The pathophysiology of these conditions is not widely understood; however, peripheral and central mechanisms appear to play a role.

Introduction

The reported prevalence of traumatically induced myofascial conditions depends greatly on the diagnostic definitions applied to them. Moreover, a traumatic etiology may not be limited to a single event (*eg,* a motor vehicle accident) [1], but may also include ongoing parafunctional muscular activities such as bruxism, which has been referred to as microtrauma [2]. Based on data from our center, painful conditions of the head and neck region can be subdivided as temporomandibular disorders (TMD) (35%), chronic headache (30%), neck pain (20%), and others (15%). Unlike most acute painful conditions, chronic head and neck pain tends to co-exist. More than 75% of patients who are referred to our center have multiple chronic pain complaints.

Epidemiology

Temporomandibular disorder is considered to be the most common head and neck complaint. Estimates of its prevalence range from 10% to 40% of the Western population [3]. More than 5% of the US population will seek advanced care for this condition at some point in their lives [4]. Although episodic in nature, this condition is most commonly found in its chronic form in women of child-bearing age [3].

Headaches as a group are the next most prevalent pain complaint, with 5% of the US population seeking medical care each year [5]. This includes all types of headache, not just chronic forms. Epidemiologic data, specifically on chronic post-traumatic headache, is lacking. However, cervicogenic headaches or tension headaches, which commonly accompany whiplash injuries [6,7], have an estimated 1-year prevalence in industrialized countries; 30% have tension headaches [8,9] and 2.5% [10] have cervicogenic headache.

Neck pain originating from trauma is less common than TMD or headache. Post-traumatic myofascial neck pain is a sequel of a whiplash type of injury in most cases. These acceleration-deceleration injuries of the cervical spine most commonly result from rear-end motor vehicle accidents; however, some forms of contact sports can also result in this type of injury. Approximately 1 million whiplash injuries occur every year in the United States [11,12]. Whiplash-associated neck injuries usually resolve quickly, with 47% of injured patients performing their normal activities within 4 weeks; only 2% continue to be absent from pre-accident activities after one year [13]. However, those patients (2%) suffering from whiplash-associated disorders (WAD) tend to have a poor prognosis and form the bulk of those patients suffering from chronic neck pain in pain clinics [13].

Pathophysiology

Myofascial pain is a descriptive term for which the pathophysiologic mechanisms are not well elucidated. Many cases of myofascial pain can be traced back to an accident or injury. Cervical whiplash injury is usually most easily associated with a traumatic event. However, TMDs and some forms of headache also occur secondary to trauma [1]. The pathophysiologic process that leads to chronic myofascial pain has not been completely described. Initially, soft tissue trauma results in local tissue deformation in muscle, ligaments, and tendons, which may result in sprain or strains and associated neurovascular disruption. The involved and surrounding muscle often responds with a clinically discernable increase in tone or frank spasm [11]. This phenomenon is thought to be integral to the pain process in myofascial tissues. A theory first proposed by Travell *et al.* [14], and subsequently added to by other authors [15,16], suggests that muscle hyperactivity leads to

fatigue, spasm, and pain, which reinforce further myo-spasm that results in a positive feedback loop. This theory is conceptually attractive and logical but has never been supported objectively [17].

In the case of short-term muscle pain (*eg,* post-exertion pain, the algesia can be explained in terms of ischemia and acidosis resulting in the release of inflammatory mediators and pain-inducing neuropeptides, which stimulate muscle nociceptors [18–20]. This mechanism may not apply the same way in chronic muscle-associated pain because of the relatively short time frame associated with the action of the inflammatory mediators involved. However, no other satisfactory alternative mechanism has been advanced [20,21]. Objective studies of chronic myofascial conditions become more complicated if the source of pain is difficult to identify. As these conditions become more chronic, the distinction between muscle pain or one of the supporting tissues becomes less clear. The actual source of pain in myofascial conditions may differ from other muscle pain conditions such as dystonias, in which pain is attributed to muscle contraction, tendon and joint tension, nerve root compression, or extremes of posture [22].

There is a void in the basic science that deals with the generation, reception, and transmission of pain in muscle [20,21] and, therefore, myofascial tissues. Much of the basic research on muscle and nerve physiology is based on animal models and inferred to humans [23]. This has led to an inability to truly justify clinical treatment modalities for chronically painful conditions (*eg,* TMD and whiplash) based on solid physiologic principles. Furthermore, it must be recognized that pain is a multi-factorial experience [24] and is different from nociception, which is considered a summation of tissue level events that allows for the identification of and reaction to noxious stimuli [23]. In muscle, the nerve fibers identified with nociception are small in diameter, slow-conducting myelinated group III, or non-myelinated group IV fibers [25]. These fibers are associated with free nerve endings that respond to a threshold stimulus within a limited receptive field [22]. They are typically located in the walls and supporting connective tissue of muscle blood vessels [27]. The dominance of one type of nerve fiber is subject to great variability among different muscles [23]. Electron microscopic studies also show different receptor subtypes and are associated with group-III and group-IV fibers [28]. It has been well demonstrated that some muscle receptors respond to one stimulus and others respond to a range of stimuli, indicating that the different types of nociceptors, which are functionally and morphologically different, may be present in muscle [23,29].

Nociceptors can be excited by many stimuli, including mechanical, thermal, and chemical [30]. Ischemia has been considered a promoter of nociceptor afferent discharge [30]. Although the exact mechanism is unknown, it is thought that a decrease in pO_2 or an increase in proton concentration is responsible for the release of secondary messengers, which sensitize the receptors [23]. These sensitizing substances, which appear to be chiefly inflammatory mediators and neuropeptides, have individual modulating effects [18,19,25]. Some agents, such as bradykinin and serotonin, can stimulate nociceptors directly and others, such as prostaglandins, facilitate pain that is triggered by other stimuli [30]. Chemical mediators such as substance P (SP) and calcitonin gene-related peptide (CGRP) are also produced by the primary afferent nerve fibers and are associated with sensitizing receptors; however, it is unknown if they influence a specific subtype of muscle receptor or if it is another mechanism through which they modulate nociceptive responses [23].

These peripheral modulation processes have potential significance in two ways. The first is that nociceptors, which normally have a threshold above a normal operating range, can be sensitized to selectively respond at a lower threshold by an inflammatory or hypoxic process [25,29,31,32]. The second is that there is evidence that neuropeptides influence the actions of cells mediating the immune response such as those involved in arthritis and other inflammatory conditions [33]. These physiochemical interactions are complex and cascade-like, producing a non-linear or self-potentiating response [23,34–36]. Therefore, local sensitization may produce conditions, which favor maintenance of nociceptor discharge with minimal stimulation. This may be a relevant consideration in conditions in which increased muscle tone is present.

The central projections of the myofascial pain pathways emanating from the orofacial region enter the trigeminal nerve (V) through the gasserian or semilunar ganglion, which is where the primary afferent cell bodies are located [30]. Similarly, upper cervical pain is passed through the roots of the dorsal horn of associated segments [37•]. The second order neurons, which are thought to be associated with nociception from the orofaclial meningeal and upper cervical regions, lie in the subnucleus interpolaris and laminae I, II, and V of the subnucleus caudalis of the trigeminal ganglion. These are stimulated by fibers that ascend and descend in the spino-thalamic tract; some may project directly to the thalamus [38,39]. Interconnections between the sensory complex and motor nerve nuclei exist, which may facilitate reflex responses [21].

Animal studies have demonstrated two main types of neurons in sub-nucleus caudalis that respond to painful stimuli. Wide dynamic range neurons (WDR) are responsive to noxious and non-noxious stimuli and nociceptive specific (NS) neurons. The WDR neurons and the NS neurons are located in the laminae of caudalis and are associated with the termination of small fiber afferent nerves from the investing fascia, jaw joint capsule, and muscles [30]. Of significance is that the WDR neurons and the NS neurons can be activated by stimulating the jaw joint and the jaw and tongue muscles, which demonstrate afferent convergence [25,26,40]. This feature may explain the poor localization of chronic pain from deep sources in the head and neck area and the pain referral pattern of specific conditions [24,30]. Intrinsic to these observations is

the concept of receptor fields. Although each nerve ending subtends a specific tissue area, the secondary neurons respond to a larger field, which is poorly defined in the case of caudalis neurons that receive deep nociceptive stimulation. Evidence from studies performed with animals suggest that receptor fields can be plastic [26]; receptor fields may enlarge and overlap as a result of central sensitization [30].

These central neuroplastic changes can alter the size and sensitivity of a receptor field to stimulation and may allow the second order neurons to become independent of peripheral nociceptive input [23,41]. Neuropeptides such as SP and *N*-methyl-D-aspartate have been implicated in the induction of neuroplastic changes [42–45].

N-methyl-D-aspartate receptor stimulation appears to increase the reflex contraction of flexor muscles and reflex facilitation to some degree [46]. However, γ motor neuron activity is likely to be reduced, unless the nociceptive stimulation is from tissue outside of the muscle (*eg,* a joint) [23]. This is significant because the link necessary to establish a self-perpetuating feedback loop with α motor neurons within a muscle is not established, thus the concept of pain leading to muscle hyperactivity (as suggested by Travell) is unsupported, at least through this mechanism.

The reception and transmission of pain from the jaw joint apparatus appears to be less ambiguous. High concentrations of free nerve endings are evident in the joint capsule, lining, and retrodiscal tissues [21,23]. These are connected to the trigeminal sensory nuclear complex by slow, small-diameter fibers [30]. A convergent association with multiple second-order neurons is then found, which appears to be subject to the same types of ascending and descending modulation as the muscle afferents [19,23,26].

Local modulators of joint pain, which have been recently studied by Kopp [47], include neuropeptide Y (NPY), serotonin, and interleukin 1 β (IL-1β). NPY appears to act as a regulator of inflammation severity in the rat model, although temporomandibular joint (TMJ) synovial serotonin levels were found to be positively correlated with pain provoked by movement. Kopp [47] also notes that IL-1β levels in synovial aspirates in patients with arthritic TMJs positively correlated with their subjective pain and tenderness to palpation.

The exact role of inflammation in the maintenance of post-traumatic myofascial pain is unknown. However, the animal data suggests that it is a key component in directly or indirectly promoting the pain process through peripheral and central mechanisms.

Diagnosis

Myofascial pain conditions represent a group of disorders that are regional in nature. This differs from diffuse conditions such as fibromyalgia [48]. The diagnosis of myofascial pain conditions is based chiefly on history and clinical findings. Taut bands, trigger points, and tender areas within the deep tissues characterize myofascial pain

conditions. However, their pathophysiologic significance in myofascial pain is unclear [49]. Travell and Simons [50] have described trigger points as tender areas within muscle that, when palpated, will cause pain in a manner consistent with the pain about which patients complained. Pain referral patterns for trigger points are well described and consistent, but do not follow strict neuroanatomic dermatomes or myotomes. Trigger points are found within the taut bands; manipulating them may cause a twitch or a local muscle contraction [50].

Objective tests for myofascial pain have been generally disappointing. Electromyography and thermography have yielded non-specific, inconsistent, or conflicting results [51–54].
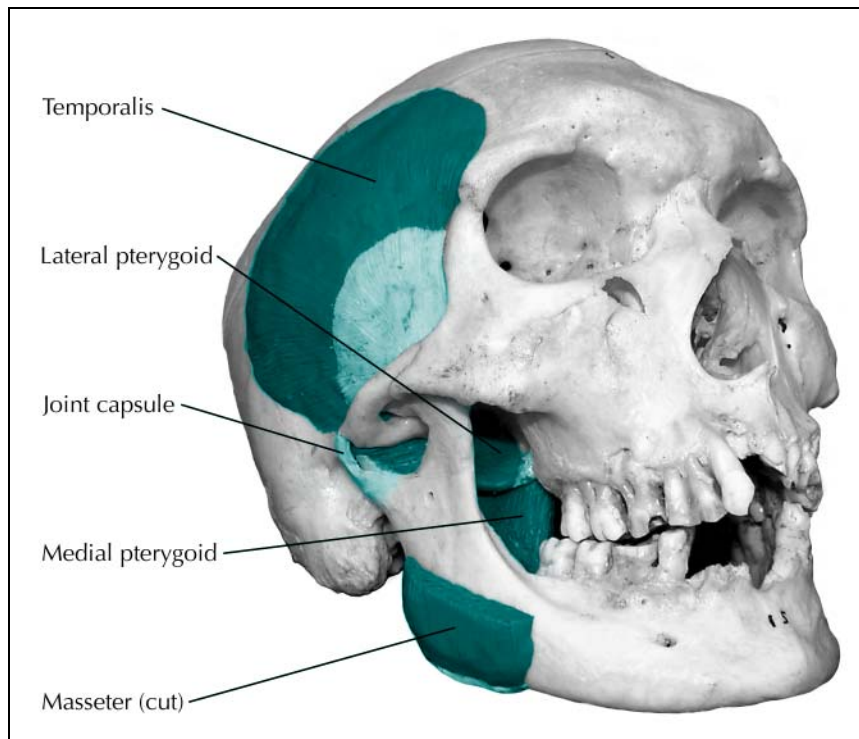
Temporomandibular Disorders

Temporomandibular disorder is a collective term used to describe a group of conditions involving the TMJ, masticatory muscles, or associated structures (Fig. 1). There has not been universal agreement regarding the pathophysiologic mechanism underlying these disorders or the most consistently effective methods of treatment [55].

Some specific etiologic factors have been proposed for TMD. These include masticatory muscle activity, trauma, psychologic factors, and systemic diseases such as arthritis [3,55–57]; the role of occlusion remains uncertain [58]. It is reasonable to suggest that TMD may be considered a multifactorial disease or an expression of similar symptomatology in different diseases.

Anatomically, the TMJ is a ball and socket arrangement in which the mandibular condyle articulates with a fossa in the temporal bone. The joint space is separated into a discrete upper compartment and lower compartment by a fibrocartilage meniscus. The joint is surrounded by a capsule, which is innervated by a branch of the temporal nerve [51]. The muscles of mastication are divided into the opening and closing muscles. Painful TMD, compared with oromandibular dystonia [59], is usually associated with the jaw-closing muscles. These are the masseter and temporalis muscles on the external surface of the mandible and temporal bone, with the medial and lateral pterygoids on the medial surface of the mandible (Fig. 1). These muscles work in concert to close and to anteriorly and laterally position the mandible. Because of this, painful parafunctional activities such as clenching or bruxing tend to cause symptomatology in the closing muscles.

Temporomandibular disorder is characterized by subjective pain and stiffness associated with the jaw joints or closing muscles. The joint pain is inflammatory in nature and is most severe during joint loading or capsular stretching. The myofascial component is exacerbated by muscular activity. Therefore, opening the mouth widely and chewing hard food usually incite TMD pain. Initiating factors may be attributed to macrotrauma (*eg,* a blow to the jaw), but result from microtrauma more commonly [2]. This mild and ongoing trauma is often the perpetuating factor for the

Figure 1. Anatomy of masticatory system showing jaw-closing muscles and cutaway of the temporomandibular joint capsule.

condition, which maintains joint inflammation and myofascial pain. This micro-trauma commonly takes the form of masticatory muscle parafunctional activity such as stress-induced clenching or bruxing.

Work has been published that examines the mechanistic similarities between TMD and other head and neck disorders. One such group of disorders is the focal dystonias. These conditions are characterized by centrally mediated increases in muscle tone that often result in spasms of a local muscular group. Common examples include torticollis, writers cramp, and blepharospasm [60]. The spasms are modified by psychologic factors (*eg*, stress) and can often be temporarily abolished with maneuvers such as sensory tricks (geste antagonist) [61]. These conditions may be centrally associated with an alteration in the distribution of dopamine receptor populations, specifically the striatal D2 receptor [62].

Lobbezoo *et al.* [63] and Lavigne *et al.* [64] have examined D2 receptor population responses in people with bruxism. The conclusion of these studies reinforces a possible role of the central dopaminergic system in regulating this disorder. Although asymptomatic bruxism is generally not considered a TMD, its rhythmic and arrhythmic form (clenching) is strongly associated with TMD.

Therapy for TMD depends on the severity of pain and dysfunction and the degree of anatomic joint derangement [65,66]. The joint component is addressed in a similar fashion to other orthopedic injuries. Initially, rest and anti-inflammatory therapy is indicated for acute joint injury and inflammatory exacerbations. Surgical intervention may be necessary for internal joint derangements to correct meniscal displacements or arthritic changes [67].

For the myofascial component, the key to successful management is control of aberrant muscular activity. Clenching, bruxing, and schedule-induced muscular hyperactivity are characteristic of TMD [68]; however, frank muscular spasm is not [65]. Orthotic appliances have enjoyed immense popularity in TMD therapy, although how they work in reducing muscular activity has not been established. There have been multiple proposed mechanisms for the positive effect on TMD seen in many patients who are treated with this modality. However, there is no evidence suggesting that any one mechanism explains an orthotic's effectiveness [69]. The orthotic appliance is generally a hard acrylic plate that covers the lower or upper teeth, opening the bite slightly. If the orthotic appliance is examined more closely, the underlying theme of orthotic therapy is sensory alteration by altering jaw position, muscle spindle length, or intercuspal relationships. Therefore, splint therapy may simply be a sensory trick affecting a clenching or grinding component of TMD in the same way that stroking the chin abolishes muscle spasm in torticollis [70]. Studies suggest that these appliances are effective in curbing pain-inducing muscular activity in approximately 60% of patients, regardless of design [69].

Pharmacologically, muscle activity can be modified with the use of centrally acting oral muscle relaxant medications such as benzodiazepines, antihistamine derivatives, tricyclic derivatives, or γ-aminobutyric acid derivatives. For many patients with TMD, this form of therapy is not totally effective in terms of the degree and duration of pain relief achieved [71,72]. This may be the result of the therapeutic agent being too weak or non-specific to be used to its full therapeutic potential. These systemic muscle relaxants have no selectivity with respect to specific groups of skeletal
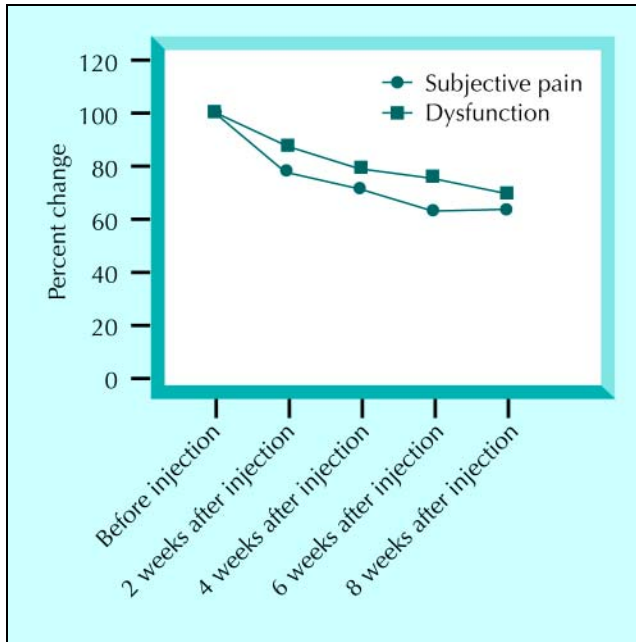
Figure 2. Relative change in mean Visual Analogue Scale pain scores and mean dysfunction index of 46 patients with temporomandibular disorder after injection of 150 units of BTX-A, which was injected bilaterally into the masseter and temporalis.

muscles and, in high doses, can have significant side effects that limit the duration and intensity of treatment.

The latest advance in TMD therapy is the use of botulinum toxin type A (Botox, Allergan, Irvine, CA), in controlling the myogenous component of these conditions. BTX-A reduces maximum contractile strength and muscular resting tone when it is injected into the masticatory muscles [73,74]. This leads to an improvement in objective and subjective signs of TMD (Fig. 2) [75,76].

Whiplash

Cervical whiplash injuries are variously referred to as acceleration-extension injury, extension-flexion injury, and cervical sprain syndrome [11]. There is limited information about the pathophysiology of cervical whiplash injuries, despite the common nature of this condition and its tremendous social impact.

Rapid extension followed by flexion of the neck is attributed to causing cervical whiplash injury. Because of its relatively high mass, the head remains behind as the shoulders move forward at the time of a rear impact. As the neck musculature begins to contract in reaction to the impact, the head is accelerated forward and may achieve forces in excess of 12 G from an impact of 30 Km/h because of its inertia [77]. The physical effect is to impart excessive stretching and compressive forces to the muscles, ligaments, and other supporting tissues within the neck [78]. The pattern of injury is further complicated by factors such as head position (rotation and flexion) at the time of impact. Computer models and cadaveric studies have dem-

onstrated the potential for injuries to hard and soft tissues [79,80].

Despite extensive studies, a characteristic cervical whiplash injury has not been identified in the neck [81,82]. Injury may also affect structures beyond the neck, which accounts for the varied presenting symptoms. For example, dysphagia may result from a retropharyngeal hematoma sustained during the whiplash or glossopharyngeal dysfunction secondary to an associated TMD.

Although most cases of WAD resolve quickly and completely [13], some patients suffer ongoing cervical pain with reduced range of motion, which is a prognostic and therapeutic dilemma.

Similar to TMD, WAD injuries have the potential to involve joints and soft tissue. The role of the zygapophysial joints in the generation of pain and dysfunction in whiplash injury has been the subject of considerable research [83]. Local anesthetic blocks of the nerves that supply these joints yield a reduction in symptoms in 50% of the patients with chronic whiplash pain who were observed [84]. These results suggest that the other 50% may suffer pathology that is related to soft tissue rather than to the joints. It has been observed that nearly nine out of 10 patients with whiplash demonstrate some degree of muscle spasm or aberrant muscular activity [6]. Such findings raise several questions. Does cervical muscular dysfunction cause ongoing excess loading of the zygapophysial joints, yielding the clinical picture of chronic whiplash? Is muscular dysfunction the body's attempt to splint a subtly injured cervical spine?

For most patients, initial treatment of whiplash-associated pain is usually pharmacologic in nature. The medications commonly prescribed are oral muscle relaxants and anti-inflammatory drugs [85]. Similar to TMD treatment, the effectiveness of these drugs is limited in some patients by their systemic therapeutic effect and unfavorable side effects. Physical therapies, such as range of motion exercises and muscle strengthening exercises, have been beneficial for pain and dysfunction on a time-limited basis. However, there is little evidence that physical therapies specifically aimed at the musculature (*eg*, transcutaneous electrical nerve stimulation, ultrasound, heat, ice, and acupuncture) are effective in improving the prognosis in acute WAD [13]. The only form of treatment that has clearly shown benefit for patients with chronic WAD is radiofrequency neurotomy. McDonald *et al.* [86] followed 28 patients with zygapophysial joint pain who were treated with percutaneous radiofrequency medial branch neurotomy. They report a response rate of 71% to complete pain relief for a median duration of 422 days [86]. However, this only addresses the 49% of patients who have symptomatology that can be attributed to orthopedic injury [84]; it leaves a considerable void in the treatment options for the majority of whiplash sufferers.

The use of botulinum toxin in the treatment of WAD is relatively new and has not been extensively studied or

Table 1. Follow-up assessments of patients with WAD 2 to 4 weeks after treatment

	*Mean total pain, saline range 0–30 (SE)	Mean total pain, BTX-A range 0–30 (SE)	Mean total ROM (in degrees), Saline (SE)	Mean total ROM (in degrees), BTX-A (SE)	Mean Vernon-Mior saline, range 0–50	Mean Vernon-Mior BTX-A, range 0–50
Pre-injection	13.3 (2.0)	16.2 (1.7)	316 (15.8)	310 (21.7)	13.7 (1.9)	18.1 (2.5)
Week 2	9.9 (1.6)	12.1 (1.4)	337 (12.7)	325 (20.1)	N/A	N/A
Week 4	14.1 (2.1)	10.0 (1.3)	308 (12.9)	343 (17.8)	12.0 (1.5)	15.2 (2.0)

*Mean total pain is derived from composite Visual Analogue Scales, with a range of 0 (no pain) to 30 (worst pain).
The Vernon-Mior Scale is a modified Obesity Back Index.
BTX-A—Botox (Allergan, Irvine, CA); ROM—range of motion; WAD—whiplash-associated disorders.

reported. This is also the case for chronic neck pain that is not associated with a whiplash injury. A pilot study conducted by the authors exploring the potential benefits of relaxing selected neck muscles with BTX-A has yielded some positive results [87]. In this randomized, placebo-controlled trial, 28 patients with a diagnosis of chronic grade II WAD [87] were injected with 100 units of Botox (Allergan Inc., Irvine CA) or saline. Each patient received five injections of 0.2 mL each into one or more of the splenius capitis, rectus capitis, semispinalis capitis, and trapezius, bilaterally. The five injection sites were chosen by palpation, and corresponded to the five most tender cervical muscular points. The injection was administered by a 30-gauge needle without electromyography guidance. Follow-up assessments were performed at 2 and 4 weeks after treatment. Three outcome measures considered were subjective pain, objective range of neck motion, and subjective function.

The treatment group showed a trend toward improvement in range of motion and reduction in pain 2 weeks after the injection (Table 1). The treatment group was significantly improved from pre-injection levels ($P < 0.01$) 4 weeks after the injection was administered. The placebo group demonstrated no statistically significant changes at any post-treatment time. The functional index demonstrated a trend toward improvement in the treatment group, but it was not enough to be significant.

These results suggest that pain relief and increased range of motion may be achieved by relaxing some of the symptomatic cervical musculature. Unpublished follow-up data on these patients for an additional 3-month post-study period showed that the treatment group continued to demonstrate significant pain reduction and increased range of motion 2 months ($P < 0.05$) and 3 months ($P < 0.05$) after the injection, but not at 4 months.

Headache

In many instances, trauma to the head and neck region result in headache. Some of these headaches are subsequent to closed head injury and cannot be attributed to myofascial pathology [88]. However, some headaches seen in conjunction with whiplash injuries and TMDs have myofascial components [89]. These are commonly tension-type headaches or cervicogenic (cervical-associated) [90] headaches. Studies examining post-traumatic headaches in relationship to the diagnostic criteria of naturally occurring headaches have found no differences [91]. This implies that trauma as an initiating event does not substantially alter defined headache types, which is seen in other circumstances.

Similar to other painful conditions of the head and neck, common post-traumatic headaches are thought to have a peripheral and central component [92–94].

The peripheral mechanism by which a tension headache is generated is not completely clear. The peripheral trigger, likely initiating the process, may be a single stimulus or multiple stimuli acting in an additive fashion. In a tension headache model system, Jensen and Olesen [95] described dental clenching as a trigger, suggesting that varying amounts of masticatory muscle activity can stimulate an acute tension headache episode in test subjects. The International Headache Society has defined one form of tension headache as involving the pericranial muscles [96]. Therefore, the increased levels of masticatory muscle activity seen in TMD may be a headache trigger. In this proposed chain of events, post-traumatic tension headache would co-exist with TMD and the temporalis muscle would be the common myofascial element.

Similarly, an association between cervicogenic headaches and whiplash injuries can be made. There is a debate regarding if the neck pathology is responsible for the headache or just associated with it [94]. However, post-traumatic cervical-associated headache was treated with BTX-A injections into the cervical musculature in a pilot study published by the authors [90]. The results showed that relaxation of the cervical muscular with BTX-A was more effective in relieving subjective headache pain than similar saline injections. This evidence is suggestive of a myofascial component to these headaches that have been triggered by a traumatic event.

Conclusions

Post-traumatic myofascial pain in the head and neck region is usually more than one condition. The pathophysiology is not

well understood, which has made treatment and prognosis difficult for the clinician. More research is needed to better identify the processes that convert an acute injury into chronic myofascial pain.

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